Recovery Kernel

DISCONTINUANCE PROTOCOL: METHAMPHETAMINE

The Crash, the Timeline, and the Road Back to a Working Dopamine System

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This guide follows the same structure as the Recovery Kernel's Forced Discontinuance series — a safety framework, a genetic cross-reference, a use-pattern classification, a recovery protocol, and a full protocol map — adapted to what methamphetamine discontinuation actually is. It is not a seizure risk the way benzodiazepine withdrawal is. It is, in the Recovery Kernel's own words, 'one of the most profoundly unpleasant biological experiences' a person can go through, and it carries a real and serious risk of its own: the crash produces a depth of depression and anhedonia severe enough to be a genuine suicide risk window. This guide treats that risk with the same seriousness the benzo guide gives to seizures.

Section 0 — Immediate Safety: Read This First

⚠ CAUTION

METHAMPHETAMINE WITHDRAWAL WILL NOT KILL YOU THROUGH SEIZURE THE WAY BENZODIAZEPINE OR ALCOHOL WITHDRAWAL CAN. BUT THE CRASH THAT FOLLOWS HEAVY OR PROLONGED USE PRODUCES ONE OF THE MOST SEVERE PSYCHOLOGICAL STATES IN ADDICTION MEDICINE, AND THE DEPRESSION IT PRODUCES IS A GENUINE SUICIDE RISK WINDOW. TAKE THE CRASH AS SERIOUSLY AS ANY PHYSICAL WITHDRAWAL DESCRIBED ELSEWHERE IN THIS LIBRARY.

Seek emergency help immediately if you experience:

- If you have any thoughts of suicide or self-harm, or feel like your depression is more than you can handle—call or text findahelpline.com, or text HOME to 741741, both available 24/7.
- Chest pain, irregular heartbeat, shortness of breath, sudden severe headache, or facial drooping/arm weakness — this can indicate a cardiac event or stroke, both real risks after heavy stimulant use, and require calling 995 immediately.
- Paranoid delusions, hallucinations, or an inability to distinguish what's real — meth-induced psychosis can persist for days to weeks after last use and requires medical evaluation.
- Extreme agitation or a feeling like you're gonna lose control of what you do.

SANCTUARY

The crash itself — extreme exhaustion, hypersomnia, intense hunger, and a wave of depression — is expected and is not, on its own, a medical emergency. It is the body

beginning to recover. The distinction that matters is the same one the SSRI guide names: mood and safety, not discomfort. If in doubt, reach out.

Section 1 — Why This Guide Exists

The Recovery Kernel's own meth guide states this plainly: 'something has not gone wrong. Your brain is recalibrating. The misery is the signal that repair is beginning.' That sentence is the organizing idea behind this entire discontinuance protocol. Meth withdrawal has no chemical antidote and no safe substitute-and-taper strategy the way benzodiazepines or opioids do — there is no equivalent of a cross-taper here. What there is instead is a well-documented, predictable biological timeline, and knowing that timeline in advance is itself a form of harm reduction: it is the difference between 'this misery means I need more meth to feel normal' and 'this misery is Day 3 of a documented process that ends.'

A Brief History: How the Danger Was Discovered

Methamphetamine first synthesized from ephedrine in 1893 by the Japanese chemist Nagai Nagayoshi, and crystallized into the form still recognizable today in 1919 by Akira Ogata. It was patented and sold in Japan in the 1920s under the brand name Philopon, marketed for fatigue, narcolepsy, and weight loss — a legitimate pharmaceutical product, not yet understood as dangerous.

That changed with its mass deployment in World War II. German chemists at Temmler patented their own methamphetamine formulation in 1937, and by 1938 it was being sold over the counter under the brand name Pervitin — distributed to Wehrmacht soldiers in enormous quantities (estimates run into the tens of millions of tablets) specifically to sustain the sleepless, forced-march pace of the Blitzkrieg, and to civilian factory workers to sustain long shifts. Japan ran a parallel program, issuing methamphetamine to kamikaze pilots and to civilian factory workers for the same reason: it let exhausted people keep functioning past the point otherwise their bodies would have stopped them.

THE CENTURION

This is the historical origin of the exact pattern in Section 5 of this guide — using a stimulant to sustain an unsustainable work pace. It was true for exhausted soldiers and factory workers in 1940, and it is true for the same pattern today.

The danger became undeniable in the aftermath. Japan in particular experienced a massive postwar epidemic once wartime military stockpiles were dumped onto the civilian market — by some estimates, a meaningful percentage of the population was misusing the drug within a few years of the war's end. The scale of addiction, psychosis, and physical breakdown this produced was severe enough that Japan enacted the Stimulant Control Law in 1951, one of the first drug-specific stimulant regulations in the world — a direct legislative response to what unrestricted access had just demonstrated at national scale. What took a global war and A national epidemic to establish in the 1940s is, in miniature, the same lesson this guide's

Section 2 timeline exists to teach in advance: this drug's capacity for harm scales directly with how much the body is asked to run on it without rest.

Section 2 — The Withdrawal Timeline

Timeline	What's Happening Biologically	What It Feels Like	What to Do
Hours 0–24 (The Crash)	Dopamine reserves depleted; receptor signalling collapsing	Extreme exhaustion, hypersomnia, intense hunger, depression, anxiety	Sleep. Eat. Hydrate. Don't fight the crash—it's the body starting to recover. Electrolytes.
Days 1–5 (The Pit)	Dopamine at floor; D2 receptors still downregulated; the system is, in effect, screaming for the chemical it adapted around	Severe depression, anhedonia (inability to feel pleasure), profound fatigue, intense cravings, disrupted sleep, cognitive fog	This is the highest relapse and highest suicide-risk window in this entire guide. Do not be alone if avoidable. Text findahelpline.com if crisis. Begin the Tier 1 supplement support (Section 8).
Days 5–14 (The Gray)	Acute symptoms resolving; dopamine system still significantly impaired; receptor recovery barely beginning	Persistent flatness, low motivation, continued insomnia, cognitive impairment, intermittent cravings	Start light exercise — this is the repair signal (Section 3). Start identifying and avoiding environmental use-triggers.
Weeks 2–4 (The Slow Return)	D2 receptor density starting to show measurable recovery; dopamine synthesis slowly picking up; prefrontal cortex repair kicking in	Mood improving gradually and unevenly; cognitive fog clearing up; cravings becoming sporadic instead of constant; sleep steadying.	Set a steady daily routine. The first achievable behavioural goals are set here, not beforehand.
Months 1–3	Measurable D2 receptor recovery; grey matter rebuilding in the prefrontal cortex; dopamine synthesis normalising	Genuine improvement with periodic cravings and mood variability; post-acute withdrawal (PAWS) episodes possible	Identity reconstruction work becomes appropriate here — not earlier. This is when capacity genuinely starts returning.
Months 3–12	Significant receptor recovery; BDNF-driven neuroplasticity	Mood near baseline for most people; cravings mostly context/cue-	Maintain every protocol below. Protect this window

Timeline	What's Happening Biologically	What It Feels Like	What to Do
	actively rebuilding damaged dopamine axons; prefrontal cortex function substantially restored	triggered rather than spontaneous; cognitive function largely restored; genuine enjoyment of life becomes accessible	from major stress events where possible — the system is rebuilding, not yet at full structural strength.

Section 3 — How Long Until the Brain Is Actually Back to Normal

This is the question underneath every stage of the timeline above, and it deserves a direct answer rather than a vague reassurance. The honest answer has two parts: the acute crisis resolves in weeks, but full receptor-level recovery takes considerably longer — and knowing the real number changes what 'normal' should be expected to feel like at each stage.

THE CENTURION

The specific data point, cited directly from the neuroimaging research the Recovery Kernel's own material draws on (Volkow et al.): dopamine D2 receptor recovery after methamphetamine cessation is significant, but substantial recovery takes 12 to 17 months of sustained abstinence. This is measurably slower than alcohol (significant D2 receptor improvement by month 1, per Heinz et al.) or cocaine — methamphetamine produces the most extensive dopamine terminal damage of the common stimulants, and the repair timeline reflects that.

Within that larger 12-17 month arc, there is a faster, more acute rebuilding window worth knowing about specifically: the first 90 days. The Recovery Kernel's neuroplasticity framework documents this in detail — days 31 to 60 are when new neural pathways begin forming and old ones weaken; days 61 to 90 are when those new patterns start to consolidate into structurally measurable change. By 90 days, with consistent exercise, sleep, and the other neuroplasticity inputs in Section 8 below, dopamine receptor density is significantly improved, gray matter volume is measurably increased, and cravings are dramatically reduced for most people — even though the fuller 12-17 month recovery is still in progress underneath that.

Recovery Milestone	Realistic Timeline
Acute crash resolves	Days 1–5
Cravings shift from constant to episodic/cue-triggered	Weeks 2–4
First measurable DRD2 receptor recovery on imaging	Weeks 2–4 through Month 1
90-day neuroplasticity window closes — new habit architecture structurally consolidated	Day 90
Mood and cognition near baseline for most people	Months 3–12

Recovery Milestone	Realistic Timeline
Substantial D2 receptor recovery (headline number)	12–17 months

BEING HEARD

If you're at month 4 and still having flat, gray days, that is not a sign the process has failed or that you're broken in some way the timeline doesn't account for. Per the data above, you are inside a genuinely still-recovering system — 'near-baseline' at months 3-12 means exactly that: closer, not finished. The finish line for the deepest layer of this repair is closer to a year and a half than a season. That's not a discouraging number. It's an honest one, and it means the flat days you're still having are expected, not evidence of failure.

A Straight Talk Disclaimer: These Timelines Assume Real Sobriety

Every number in this section — the weeks, the months, the 12-17 month headline figure — was measured in people who stopped methamphetamine and stopped there. In practice, a lot of recovery doesn't look that clean. Two substitutions are common enough to name specifically, because each one interacts with the exact repair mechanisms this section describes, and each one plausibly slows the clock rather than being a neutral bridge through it.

Replace	What It Actually Does to the Recovery Mechanism	Likely Effect on the Timeline
Caffeine (used to manage the fatigue and low-motivation phases in Section 2)	Caffeine doesn't deplete dopamine like methamphetamine does, so it isn't 'the same thing in miniature.' But it does stimulate the HPA axis — adding cortisol-system activation on top of a system, per Section 6, that may already be running hot (especially in the Met/Met 'Worrier' profile) — and heavy or late-day caffeine use directly interferes with sleep quality and duration. Section 8's Tier 3 is explicit that sleep is when BDNF synthesis and dopamine-system repair peak.	Plausibly prolongs the timeline indirectly, by undermining the sleep quality the actual repair work depends on, rather than by any direct dopamine mechanism. The more caffeine is substituted specifically to power through the fatigue Section 2 describes, the more it's fighting the exact process that fatigue is part of.
Alcohol (used to fall asleep)	This is the more concerning substitution of the two. Alcohol is sedating, but it fragments sleep architecture and specifically suppresses REM sleep — and REM is, per this library's own material, exactly the sleep stage where 'emotional memory processing and dopamine system maintenance	Plausibly prolongs the timeline directly, through degraded REM sleep, and introduces a second risk this section's numbers don't account for at all: a new dependence forming on top of the one being recovered from —

Replace	What It Actually Does to the Recovery Mechanism	Likely Effect on the Timeline
	occur.' Using alcohol to force sleep onset while suppressing the sleep stage recovery most depends on is close to solving the visible problem (can't fall asleep) while worsening the invisible one (the sleep that was supposed to repair the dopamine system isn't actually happening).	genuinely likely for the DRD2 A1 allele / Reward Deficiency Syndrome profile in Section 6, where the reward circuit is already primed to over-attach to whatever substance is reliably available.

⚠ **PERHATIAN**

Neither of these substitutions is framed here as a moral failure — reaching for caffeine or a drink to get through an unbearable stretch is an extremely understandable response to real suffering. But the honest version of this guide has to say plainly: neither one is a neutral bridge through the timeline in the table above. If sleep is the problem, the non-alcohol tools in Section 8's Tier 3 (melatonin, magnesium glycinate, glycine, consistent schedule) are working with the repair mechanism instead of against it.

The Fracture: Why 'A Year Off, Then I'll Be Fine' Isn't Quite Right

There's an idea worth addressing directly, because it's a reasonable-sounding conclusion to draw from Section 3's own numbers, and it's not entirely wrong — which is exactly what makes it dangerous. The idea: 'if D2 receptors substantially recover by 12-17 months, I could take a year off, let my brain heal, and then know how to use safely again, or resume with a clean slate.' The old addiction-treatment framing — 'the moment you pick up again, you're instantly back to full addiction' — isn't 100% true. But the instinct that a long break resets you to zero isn't 100% true Either one. The honest answer sits in between, and a fracture is a genuinely useful way to picture why.

What's True (Like a Healed Fracture)	What Else Is True (Why The Gait Still Changed)
The bone does heal. D2 receptor density, per Section 3's data, does genuinely recover on a real, measured timeline — this is not a euphemism or a hopeful story, it's what the imaging shows.	A healed bone is not identical to the unbroken one. It remodels around the injury. It can bear real weight again — but not automatically the same load, at the same angle, the same way, without the body having to relearn how.
Tolerance is genuinely lost during real abstinence — this is well-documented pharmacology, not guesswork.	Lost tolerance means a system that used to handle a given dose can no longer handle that same dose safely. Returning to a 'known' amount after a real break is dosing a body whose actual capacity has changed, even though the memory of what used to feel normal hasn't. This is precisely the mechanism behind

What's True (Like a Healed Fracture)	What Else Is True (Why The Gait Still Changed)
	the elevated overdose and cardiac-event risk (Section 0) seen specifically in relapse after a period of abstinence — not because the drug got stronger, but because the body's capacity to handle it got smaller while the expectation didn't.
The old 'instant relapse to full addiction' framing overstates how fixed and permanent the neurology is.	It's not baseless, either. Section 6's epigenetic material describes how reward-circuitry gene expression shifts with repeated use — and those shifted pathways don't necessarily need to be rebuilt from nothing to reactivate. A previously-carved path can be walked again faster than a new one gets carved the first time. This is why relapse after a break often escalates in severity faster than the original use did, even when baseline receptor measures have genuinely recovered.

⚠ PERHATIAN

To be plain: a period of real abstinence is not a reset button, and it's not useless either — both of the framings the introduction to this section named are partly right and partly wrong at the same time. What it actually produces is a system that has repaired in the ways Section 3 describes, while remaining structurally different from the one that existed before use ever started — different tolerance, different vulnerability to fast reactivation, different cardiovascular capacity. Trying to put the exact same weight on it, the exact same way, is where the danger concentrates. This isn't a technical Note. It's the reason relapse after a real break carries its own distinct, elevated risk profile rather than being either 'safe now' or 'instantly back to square one.'

Popular Claim Worth Correcting: 'Your Cells Fully Replace Every 7 Years'

This idea circulates widely, including inside recovery communities specifically — but it doesn't hold up to what's actually been measured, and the corrected version is more useful than the myth, not less. Different tissues turn over at wildly different rates: skin and gut lining in days to weeks, fat cells at roughly 10% per year, the skeleton over roughly a decade. But the tissue that matters most for everything in this guide — neurons in the cerebral cortex — are not meaningfully replaced at all after early development. The neurons involved in the reward pathways this entire guide describes are, mostly, the same physical cells you had before you ever used methamphetamine. They are not swapped out for fresh ones on any timeline, seven years or otherwise.

So what about age and time? Two real things, distinct from the myth. First, what recovers on the timelines in this guide is not new cells replacing old ones—it's the

density, sensitivity, and expression of receptors on the neurons that were already there, plus the synaptic connections between them (Section 6's epigenetics material). Second, aging does add a genuine complicating layer, through a different and well-documented mechanism: cellular senescence. As the body ages, some cells—including supporting glial cells in the brain—stop dividing but remain metabolically active, secreting inflammatory signaling molecules that accumulate over time and contribute to background neuroinflammation. This is real, current addiction and aging science, and it means the recovering system isn't a fixed target — it's aging in the background at the same time it's repairing, which can plausibly make the whole process feel harder or slower the more time and the more cumulative use has passed, independent of the dopamine-specific timeline in Section 3.

BEING HEARD

because the same neurons persist rather than getting replaced, the old pathways this guide has discussed — the fast reactivation risk, the learned cue responses — are not gone after a year off. They're still physically present, in the same cells, just quieter. That is exactly why writing off a year of real work as 'it didn't work' or 'I wasn't strong enough' or reaching for 'once an addict, always an addict' is the wrong conclusion to draw from a relapse — but so is assuming a year was ever going to erase pathways that were never going to be deleted in the first place, only outcompeted. The actual task was never full erasure. It's building new, competing pathways strong enough that the old ones stop being the path of least resistance — which is precisely why Section 11's Protocol 20/C-28 material talks about redirecting this exact reward circuitry toward genuine, high-intensity natural alternatives, rather than asking the circuitry itself to disappear. And it's worth saying plainly: returning to a substance after time away is not always someone deciding they could 'get away with it.' Ordinary human curiosity — wondering, testing, simply being drawn to something the way people are drawn to things — is a real and understandable pull on its own, without needing a story about willpower or moral failure attached to it.

Section 4 — What You'll Most Likely Experience

The timeline in Section 2 tells you what's happening biologically and roughly when. This section is different — it's an attempt to actually describe what it feels like from the inside, in more depth than a table row can hold, so that when it happens, you recognize it rather than panicking at it.

Anhedonia — the flattest word for the hardest symptom

Anhedonia gets mentioned almost clinically in most withdrawal material — 'inability to feel pleasure' — as if that fully captures it. It doesn't. What it actually feels like, for most people, is closer to watching your own life through glass: food doesn't taste like much, music you used to love registers as sound without the feeling attached, a joke lands as information rather than something funny, a person you love doesn't produce the warmth being near them used to produce. It is not that you've stopped caring. It's that the machinery that used to translate 'good thing happening' into

'felt good' is running at a fraction of its normal signal, because that machinery is the exact dopamine system methamphetamine hijacked and depleted. This is genuinely one of the most disorienting parts of the entire process, because it can produce a second-order fear on top of the flatness itself — 'what if I never feel anything again' — and that fear is worth naming directly: per Section 3's data, this is a documented, temporary state with a known recovery arc, not a permanent personality change.

Other symptoms worth naming plainly

- Hypervigilance and a racing, unsettled quality even through the exhaustion — the nervous system doesn't downshift as fast as the body needs sleep.
- Intense, specific cravings that come on without much notice, often set off by something totally normal—a smell, a song, a text ping—and which at that moment feels like the only thing that can make things bearable.
- Mood swings and a short fuse that can surprise you and the people around you, especially in the Pit phase (Days 1-5).
- Real cognitive fog — word-finding difficulty, trouble following conversations, a sense of thinking through mud. This is not a sign of permanent damage; it tracks the same recovery arc as everything else in Section 2 and 3.
- Vivid, sometimes disturbing dreams once sleep starts returning — the brain doing a large amount of catch-up REM processing after a period of suppressed or fragmented sleep.

This one worth a closer look, because it often the most confusing symptom rather than the most obviously distressing one. The same BDNF-driven synaptic strengthening that's repairing the dopamine system (Section 3) works partly through glutamate signaling — glutamate and NMDA receptor activity are a normal, necessary part of how synapses actually get stronger, not purely a toxic byproduct. So the intensified dream activity in this recovery window best understood as dual causation: real repair work happening, with unusually vivid and frequent dreaming as a side effect of that work, not primarily a sign of ongoing damage. For some people this goes further than just vivid — dreaming intensely enough to physically wake up from it, sometimes repeatedly through the night, which becomes its own sleep-disruption problem layered on top of the insomnia already present. Others notice they're suddenly recalling dreams in detail they normally wouldn't — waking up and being able to describe a dream at length, sometimes catching themselves talking about it as though it were something that actually happened, before the distinction settles back in. Both of these are consistent with the REM rebound and synaptic remodeling happening during this window, not signs that something Has gone wrong with your grip on reality.

BEING HEARD

If you read the anhedonia description above and thought 'that's exactly it, and I didn't have a word for it' — that reaction is common, and it's worth pausing on. A lot of people go through this particular symptom feeling like something is uniquely wrong with them, specifically because nobody described it in enough detail for them to recognise it as a

known, documented thing that happens to almost everyone going through this. It has a name. It has a timeline. It is not you.

What Makes This Harder: The Things Worth Avoiding On Purpose

Some of what happens during this recovery is going to happen no matter what — the timeline in Section 2 and Section 3 is largely fixed biology. But some of what makes the experience feel unbearable enough to drive someone back to using isn't the underlying repair process itself — it's specific, avoidable things stacked on top of it that make an already-hard window meaningfully harder. Knowing which is which matters, because it's easy to mistake 'this got worse because of what I ate/drank/didn't sleep' for 'this recovery isn't working,' when the actual answer is often much more fixable.

What Makes It Worse	Why
□□□□□□□□□□	Blood sugar spikes and crashes make the mood swings and energy dips worse, which are already happening because of low dopamine—a crash on top of a crash.
Caffeine and alcohol	Covered fully in Section 3 above — caffeine indirectly, through sleep interference and HPA-axis stimulation; alcohol directly, through REM suppression on top of the REM rebound already happening.
Nicotine and other stimulants	The obvious one, but worth stating plainly: any stimulant, including nicotine, keeps the same reward and arousal circuitry engaged that this entire recovery is trying to let settle.
Poor sleep hygiene — using screens late at night, irregular bedtime	directly undermines the sleep-dependent repair mechanism (BDNF synthesis, memory consolidation) that Section 8's Tier 3 identifies as the primary driver of the recovery timeline in Section 3.
Dehydration and skipping meals	The brain doing this much repair work needs consistent glucose and hydration; running on empty adds a second, unnecessary layer of depletion on top of the biological one already happening.
Extended isolation	Not a food or substance, but worth including here — isolation measurably worsens every withdrawal syndrome this library documents, and it removes the support contact that Section 0 identifies as protective during the highest-risk window.

□ SANCTUARY

Exercise is the clear exception to this list, and deserves to be named as one rather than lumped in with caution about overdoing things. Per Section 8's Tier 3, it is the primary mechanism generating the BDNF that drives the entire recovery timeline in Section 3 — more of it, not less, is the general direction to move in as capacity allows.

BEING HEARD

Here's the part worth saying directly: almost everything painful in this recovery — the flatness, the fatigue, the intense dreams, the cravings — is your body actively repairing itself. It is not a sign that something is wrong. It's easy, in the middle of it, to mistake the discomfort of repair for evidence that recovery isn't working, and to reach for the drug specifically to make an idiopathic-feeling wave of symptoms stop — not because the craving itself got stronger, but because nobody told you that what you were feeling was expected, temporary, and productive. Knowing that in advance doesn't Make the discomfort disappear. But it changes what the discomfort means, and that changes what you do about it.

Section 5 — The Work-Driven Use Pattern and the Dream-Wake Overlap

Not everyone who develops methamphetamine dependence started from a party or a crisis. A real and common pattern is use that began, or escalated, specifically to work — long shifts, multiple jobs, caregiving on top of a full workday, the kind of chronic overextension where stopping doesn't feel like an option. Methamphetamine's stimulant effect can look, for a while, like the only way to keep meeting a schedule that was never sustainable to begin with. This pattern deserves its own mention because it produces a specific and distinct symptom that other descriptions of meth use often miss entirely.

⚠ PERHATIAN

Chronic, severe sleep deprivation — the kind that develops from using a stimulant to push through the sleep the body was supposed to get — can produce a phenomenon where dream content begins intruding into waking perception: brief, vivid, dreamlike images or sensations while you are ostensibly awake, a sense of unreality, or moments where you can't quite tell whether something happened or you dreamed it. This is a well-documented consequence of severe sleep deprivation itself (sometimes called hypnagogic intrusion or microsleep with dream content), not a sign of psychosis on its own — though it shares some features with psychosis risk mentioned in Section 0, and any version of this that includes paranoid delusions or a break from reality testing (rather than brief, dreamlike perceptual intrusions) should be evaluated the same way Section 0 describes.

If this pattern applies to you, the recovery task includes something the rest of this guide doesn't fully cover: actually resolving the underlying overwork, not just the substance. Rebuilding a dopamine system (Section 3) while returning to the same unsustainable schedule that helped create the dependence in the first place is rebuilding into the same conditions that broke it. This is worth naming honestly rather than leaving implied.

Section 6 — Genetic Cross-Reference

Gene	What It Governs	Relevance Here
COMT	Dopamine clearance speed	Val/Val ('Warrior,' fast clearance) is the

Gene	What It Governs	Relevance Here
(Val158Met)		primary vulnerability profile for methamphetamine specifically — the fast-clearing, intensity-seeking system found in meth's extreme dopamine signal the first experience that ever fully satisfied it. Recovery requires genuine high-intensity alternatives, not just abstinence.
DRD2 / ANKK1 (Taq1A, A1 allele)	DRD2 receptor density	The A1 allele carrier's reward circuit runs with reduced baseline D2 density — Reward Deficiency Syndrome. This is the single most important genetic target for recovery supplementation (the 'Brain Stack' in Section 8), since it's the receptor system itself that needs to rebuild density, not just clear the drug.
MAOA (uVNTR)	Dopamine, serotonin, and norepinephrine are neurotransmitters that play crucial roles in regulating mood, arousal, attention, and motivation. While they all function as chemical messengers in the brain, their mechanisms of action, primary pathways, and typical associated functions differ. Here is a breakdown of each: ### Dopamine (DA) * **Primary Role:** Often associated with the brain's reward system, motor control, and executive function. It drives motivation and pleasure. * **Key Pathways:** Nigrostriatal pathway (motor control), Mesolimbic pathway (reward/pleasure). * **Function Breakdown:** * **Motor Control:**	Low-activity alleles let monoamines accumulate; high-activity alleles deplete them rapidly. The second pattern is associated with greater impulsivity and use severity — relevant to both supplement timing and relapse-risk awareness.

Gene	What It Governs	Relevance Here
	Essential for smooth, coordinated muscle movements. Deficiencies are linked to Parkinson's disease. * **Reward & Motivation:** Involved in the anticipation and receipt of rewards (e.g., food, social interaction, drugs). * **Attention/Focus:** Plays a role in goal-directed behaviour. * **Deficiency Effects:** Can lead to apathy, lack of motivation, tremors, or difficulty initiating actions. ### Serotonin (5-HT) * **Primary Role:** Often linked to feelings of well-being, mood stability, sleep regulation, and appetite control. It is widely distributed throughout the body. * **Key Pathways:** Affects nearly every major brain region involved in emotional processing. * **Function Breakdown:** * **Mood Regulation:** Stabilising mood and reducing anxiety. Low levels are often implicated in depression. * **Sleep Cycle:** Helps regulate the sleep-wake cycle. * **Appetite & Digestion:** Influences gut motility and feelings of fullness. * **Deficiency Effects:** Can manifest as irritability,	

Gene	What It Governs	Relevance Here
	anxiety, insomnia, or changes in appetite/mood swings. ### Norepinephrine (NE) / Noradrenaline * **Primary Role:** Primarily associated with the body's "fight-or-flight" response, alertness, and vigilance. It is a key component of the sympathetic nervous system. * **Key Pathways:** Involved in arousal systems throughout the brainstem and cortex. * **Function Breakdown:** * **Arousal & Alertness:** Increases heart rate, blood pressure, and general state of readiness. * **Focus/Vigilance:** Helps maintain attention during stressful or demanding situations. * **Energy Mobilisation:** Prepares the body for immediate physical action. * **Deficiency Effects:** Can lead to feelings of fatigue, sluggishness, difficulty concentrating, and low energy levels. *** ### Summary Comparison Table | Neurotransmitter | Primary Association | Key Function(s) | Deficiency Symptom	

Gene	What It Governs	Relevance Here
	Example | | :--- | :--- | :--- | :--- | | **Dopamine** | Reward, Movement | Motivation, Pleasure, Motor Control | Apathy, Tremors | | **Serotonin** | Mood, Well-being | Emotional Stability, Sleep, Appetite | Irritability, Depression | | **Norepinephrine** | Alertness, Stress Response | Vigilance, Fight-or-Flight, Energy | Fatigue, Sluggishness |	
BDNF (Val66Met)	Neural repair and plasticity signalling	Met carriers secrete less BDNF in response to activity. Exercise-driven BDNF increase is the primary neuroplasticity tool in this entire guide — it works regardless of genotype, but Met carriers may need higher exercise intensity to generate an equivalent repair signal. This is not optional in this recovery — it's the repair mechanism itself.
CYP2D6 / CYP2B6	Metabolism of certain recovery-relevant medications (e.g., bupropion)	If any medication is introduced during recovery — poor metabolizers risk toxicity at standard doses; ultra-rapid metabolizers may get no effect. Worth disclosing to a prescriber before starting anything.

How COMT Status Actually Changes What the Crash Feels Like

The COMT row above names the genotype; this is about what it actually looks like day to day, because Val/Val and Met/Met withdraw differently enough that it's worth describing separately rather than leaving it as an abstract label.

COMT Profile	Baseline Before Meth	How the Crash Manifests
Val/Val ('Warrior') — cepat clear dopamine, turunkan baseline dopamine tone	Selalu rasa kurang rangsangan bila rehat; tertarik pada situasi yang intensiti tinggi, baru, dan berisiko walaupun sebelum guna apa-apa bahan.	The crash tends to present as profound flatness and boredom more than anxiety — a kind of gray, understimulated emptiness. Motivation loss is often the most prominent complaint. Cravings frequently centre on wanting to feel anything intensely

COMT Profile	Baseline Before Meth	How the Crash Manifests
		again, not specifically on escaping distress.
Met/Met ('Worrier' / 'Sage') — slow dopamine clearance, higher baseline dopamine and stress-chemical tone	Often feels anxious or overthinks even when resting, even before using anything; the nervous system tends to be running 'hot.'	The crash tends to present with more anxiety, rumination, and agitation layered on top of the flatness — not just low mood but a wired-and-tired quality. This profile is also more prone to the hypervigilance described in Section 4, and may find the sleep-deprivation effects in Section 5 hit harder given an already-elevated stress-chemical baseline.
Heterozygous (Val/Met)	A middle ground between the two profiles above.	Often a mix of both patterns — some flat, some anxious — without either being as severe as the homozygous profiles.

GABRA2 — The 'Brakes' System and Why Stopping Isn't Equally Easy for Everyone

Methamphetamine itself not a GABA-A drug like benzodiazepines—but GABRA2 status still matters here, for a different reason. GABRA2 governs the density and sensitivity of the brain's primary inhibitory ('braking') system—the general capacity to apply the brakes on impulse, urge, and behaviour once it's already in motion. This is directly relevant to how hard 'just stop' actually is for a given person, independent of willpower.

GABRA2 Profile	What It Means for Stopping
Lower-risk / typical GABRA2 function	More baseline inhibitory 'brake' capacity available — the impulse to use, once triggered, is somewhat easier to interrupt before it becomes an action.
High-risk GABRA2 variant (thinner baseline inhibitory tone)	Less braking capacity available before use ever starts — meaning the gap between craving and action is genuinely shorter and harder to widen through willpower alone. This is a hardware difference, not a character difference, and it's the same GABRA2 architecture documented in the Recovery Kernel's GABA Braking Systems material for benzodiazepines and alcohol — it applies here too, just to impulse control generally rather than to a GABA-A drug specifically.

SANCTUARY

If stopping has felt harder for you than it seemed to be for someone else you know who used the same drug the same amount — that difference may be real hardware, not a gap in effort or commitment. GABRA2 status is exactly the kind of thing that makes 'just stop' a wildly different task for two different nervous systems facing an identical craving.

Beyond the Genes You're Born With: Does Meth Use Change Gene Expression?

Everything in the table above concerns the genetic hardware someone is born with. A separate and increasingly well-studied question is whether methamphetamine use itself changes how genes are expressed — not the underlying DNA sequence, which use does not alter, but the epigenetic marks that determine which genes get switched on or off, and how strongly. This distinction matters: nobody's genetic code is rewritten by using a substance, but the activity level of specific genes — particularly the dopamine-pathway and stress-response genes already discussed throughout this section — can genuinely shift with sustained use.

What's well-established in addiction neuroscience broadly (this is not meth-specific research alone, but methamphetamine is included in the broader literature it's drawn from): repeated stimulant exposure drives the accumulation of a transcription factor called ΔFosB in the brain's reward circuitry, which in turn alters the expression of a range of downstream genes involved in reward sensitivity and craving — this is one of the most replicated findings in addiction molecular biology, described in the foundational literature this compendium's own bibliography cites (Nestler, 2001, on the molecular basis of long-term plasticity underlying addiction). Whether methamphetamine produces additional, drug-specific epigenetic signatures beyond this general mechanism — and whether any of these changes could be passed on transgenerationally, as some animal research on other stressors and substances has suggested — is a genuinely active and not yet fully settled area of research. It would be overstating the science to present that second part as confirmed; it's an open question worth watching, not a documented fact yet.

SANCTUARY

This same epigenetic mechanism runs in both directions. The Recovery Kernel's own material on exercise-driven neuroplasticity describes 'the epigenetic feedback loop' directly — deliberate, matched physical activity produces measurable changes in gene expression on a documented timeline (habit and identity shift by months 1-3, measurable epigenetic changes by months 3-6, a new neurological baseline by month 6 and beyond). Whatever gene-expression changes use produced, the Tier 3 recovery work in Section 8 is not just symptom management — it is working through the identical biological channel, in the opposite direction.

Who This Guide Cannot Make Safe: The Genetically Contraindicated Profile

Almost everything in this guide assumes that recovery, done well, is available to anyone regardless of genetic profile — the timeline may be harder or easier depending on the hardware, but it's available. There is one profile where that assumption doesn't hold, and it's worth naming plainly rather than leaving it implied inside a genetics table.

SEVERE RISK

For one specific genetic and psychiatric combination, methamphetamine use carries a risk that is different in kind, not just degree, from everything else in this guide — including a real risk of triggering a psychotic disorder that does not fully resolve with abstinence. This is described in full below.

Profile	Why This One Is Different in Kind, Not Just Degree
COMT Met/Met ('Sage'/'Worrier' — slow dopamine clearance, high baseline dopamine and stress-chemical tone) combined with a personal or family history of psychosis, schizophrenia, or bipolar disorder	This is the same logic the Recovery Kernel's own material applies to the most potent synthetic cathinones (MDPV, alpha-PVP): a pure dopamine-flooding stimulant, delivered into a system that already runs high baseline dopamine tone and lacks serotonin's tempering influence, produces the highest documented psychosis-precipitation risk in the stimulant category. For most people, stimulant-induced psychosis — while serious — resolves with abstinence over days to weeks. For this specific combination, the risk is meaningfully different: precipitating a first psychotic episode in someone with an underlying vulnerability can trigger a condition that does not fully resolve when the drug clears, because the The drug might have triggered an underlying disorder instead of being the sole cause of a temporary state.

How to Spot This Before It's Too Late

Full genetic testing (COMT rs4680, DRD2/ANKK1 Taq1A) is the most precise way to know, but it isn't accessible to everyone, and the more urgent and available screening tool is a plain history — this is worth going through honestly, ideally before any use, or as early as possible into recognizing a use pattern:

- Any family history of schizophrenia, bipolar disorder, or a psychotic episode of any kind — in a parent, sibling, or grandparent?
- Have you personally ever had an experience, on any substance including alcohol or cannabis, where you didn't just feel intensely different but lost your grip on what was actually real — not just paranoid or anxious, but genuinely uncertain what was happening around you?
- You tend to feel overwhelmed, anxious, or overstimulated by normal levels of stimulation even without any substances—the Met/Met 'Worrier' pattern described in Section 6 above?
- Ever had a psychiatrist or doctor raise the possibility of a mood or psychotic-spectrum disorder, even one you didn't follow up on or disagreed with?

⚠ PERHATIAN

Any combination of a 'yes' on the family or personal history questions with the Met/Met stimulant-sensitivity pattern is worth treating as a genuine contraindication, not a risk factor to weigh against the appeal of the drug's effects. This is the one place in this entire guide where the message isn't 'here's how to reduce the danger' — it's 'this specific combination is why the danger can't be fully reduced.' If this describes you or

someone you're trying to help, a psychiatric evaluation before any further use — not after a crisis — is the responsible next step, and it's a "I want to understand my personal psychosis risk before I use this again."

Phenotyping Without Test Results

- Stimulants make you feel normal, focused, and alive in a way nothing else ever did? → Likely Warrior COMT profile with a DRD2 deficit underneath.
- Used stimulants specifically to perform — work harder, longer, achieve more? → Likely Val/Val COMT.
- Start with a prescription stimulant and escalate from there? → Likely a DRD2 deficit with tolerance development.
- Also struggle with depression or emotional flatness even when not using? → Likely MTHFR involvement — methylation support (5-MTHF) is a priority alongside everything else in Section 8.

Section 7 — Use Pattern Classification

Pattern	Typical Profile	Withdrawal Severity	Recommended Posture
Low	Occasionally, non-binge use; by mouth or occasional snorting; used for only a few weeks in total	Lower — crash is real but typically brief and less severe	Stopping it by yourself is fine, but having a support contact is still good idea considering the mood dip. Section 0's safety signs still apply.
Medium	Regular use, not in extended binges; weeks to a few months of use	Moderate — expect the full crash and pit phases in Section 2's timeline	A support structure and the Section 8 supplement stack from day one meaningfully reduce severity.
High	Binge-crash cycling — multi-day use periods followed by extended crashes — for months	High — the binge-crash pattern itself accelerates dopamine system depletion beyond what steady use produces	Strongly advise getting medical support during the crash phase, especially for the suicide-risk window in Days 1–5. Don't go through this alone.
Chronic / IV or smoked route, long-term	Months to years of use, especially via smoking or injection (faster, more	Highest — this is the population the 12–17 month D2 recovery	Strongly recommend full medical and

Pattern	Typical Profile	Withdrawal Severity	Recommended Posture
	intense dopamine spikes and correspondingly deeper depletion)	timeline in Section 3 was measured in	psychiatric support during the acute crash. This is also the population most likely to benefit from formal treatment engagement rather than a purely self-directed protocol.

Section 8 — The Recovery Protocol

⚠ PERHATIAN

Nothing here treats suicidal ideation, psychosis, or a cardiac event. If any Section 0 sign is present, that takes priority over this protocol.

Tier 1 — Biological Support

Two additions below speak directly to the glutamate side of this recovery, alongside NAC — meth's dopamine surge co-releases glutamate in cortico-striatal circuits, and excess NMDA receptor activation from that glutamate is a real contributor to the neurotoxicity in Section 3, and plausibly to the intensified, sometimes disturbing dreams that show up as REM sleep rebounds once real sleep returns (Section 4).

Support	Role
Tyrosine (500-1000mg, pagi, jauh dari protein)	Dopamine precursor — start once eating regularly, usually Day 3-5.
NAC (600mg twice daily with food)	Dual-action: glutamate homeostasis and craving reduction via the cystine-glutamate exchange system, AND a glutathione precursor supporting liver detoxification — chronic meth use, especially combined with any adulterants in the supply, places real burden on hepatic detox pathways, and NAC replenishes the liver's primary antioxidant molecule.
NAD+ (nicotinamide adenine dinucleotide)	Chronic substance use significantly depletes NAD+, a coenzyme central to cellular energy production and DNA repair — part of why early recovery involves such profound fatigue. IV infusion is the most effective and most studied delivery route for acute withdrawal support specifically, with some clinical programs reporting meaningful reduction in withdrawal discomfort; nasal spray, sublingual, and oral precursor (NMN/NR) forms are more accessible but less direct. This is a genuine option, not a first-line necessity — the foundational work below

Support	Role
	(sleep, exercise, nutrition) accounts for the large majority of physiological recovery; NAD+ and other advanced interventions are a real but a smaller addition on top of that foundation, not a substitute for it.
Calcium D-glucarate (200-400mg sehari)	A complement to NAC's role above, working on a different part of liver detoxification: it inhibits beta-glucuronidase, an enzyme that can reverse the liver's Phase II conjugation process and let already-packaged toxins and hormone metabolites get reabsorbed back into circulation instead of properly excreted. Where NAC supports the glutathione conjugation pathway directly, calcium D-glucarate helps make sure what the liver has already processed actually leaves rather than recirculating — relevant background support during a period when the liver is managing both the substance itself and whatever adulterants were in the supply.
Magnesium glycinate (300-400mg elemental, malam)	Magnesium sits directly inside the NMDA receptor channel and physically blocks excess calcium influx unless the cell is sufficiently depolarized — it is the body's own endogenous regulator of exactly the glutamate/NMDA overactivation described above. This is in addition to, not instead of, its role in Section 8's sleep support below.
P5P (pyridoxal-5-phosphate — the active form of vitamin B6, 20-50mg daily)	P5P 是谷氨酸脱羧酶（GAD）必需的辅因子，该酶将谷氨酸转化为 GABA。如果没有足够的 P5P，这种转化效率就会降低，导致相对更多的谷氨酸和较少的抑制性 GABA 平衡——这与本节描述的富含谷氨酸的情况直接相关。如果担心甲基化问题，请使用活性 P5P 形式而非标准 B6（吡哆醇），参考下方的 MTHFR 行。
The 'Brain Stack' — Uridine + DHA + CDP-Choline	Directly targets D2 receptor density upregulation — the structural recovery of the dopamine system itself, not just symptom relief.
5-MTHF (methylated folate)	Non-negotiable foundational methylation support, especially important if the MTHFR-linked depression/flatness pattern in Section 6 applies.
Electrolytes and hydration, protein-rich food as appetite returns	Directly supports the crash phase in Section 2; the body's nutritional depletion after a binge is real and worth taking as seriously as the psychological symptoms.

⚠ PERHATIAN

On ketamine specifically, since it comes up naturally alongside glutamate: there is genuine research interest in ketamine for stimulant use disorder, working through NMDA-related synaptic plasticity rather than by 'reversing' toxicity directly, similar to its mechanism in treatment-resistant depression. It is Emerging-tier evidence, specialist-supervised only, and worth real caution rather than pursuit here specifically — the same Reward Deficiency Syndrome vulnerability discussed throughout Section 6 that made

meth so compelling can make a second dissociative substance compelling too. Route of administration matters a real amount here: IV ketamine and esketamine (Spravato, intranasal, FDA-approved for treatment-resistant depression — an off-label use if applied to stimulant use disorder specifically) have far higher and more reliable bioavailability than oral ketamine, including troches — oral routes lose significant drug to first-pass liver metabolism, producing a less predictable effect. In this early, still-actively-repairing window, the more reliable IV or intranasal routes are generally considered the more clinically worthwhile option if this path is pursued at all; oral ketamine is more commonly discussed as a later-stage option, closer to the one-year mark once the acute dopamine and glutamate repair work in Section 3 is substantially further along. This is a conversation for a psychiatrist familiar with Both stimulant use disorder and ketamine's research base, not a self-directed addition to this stack.

Tier 2 — Medical and Psychiatric Support

- No FDA-approved medication specifically for methamphetamine withdrawal — unlike the benzo or SSRI guides, this tier is about monitoring and support rather than a pharmacological bridge.
- Psychiatric evaluation during the crash phase is appropriate if depression is severe, given the genuine suicide risk window in Days 1-5 — this is not an overreaction, it's a proportionate response to a documented risk period.
- If a prescriber introduces any medication (occasionally bupropion is used off-label for stimulant use disorder), disclose CYP2D6/CYP2B6 status if known, per Section 6.

Tier 2.5 — 'Ghost Scaffolding': Psychiatrist-Supervised Stimulant Stabilization

This deserves its own honest treatment rather than a single bullet, because it's a real approach that exists in specialist addiction medicine, and it's also genuinely easy to misunderstand as 'just get a prescription for the same kind of drug.' It is not that, when done correctly — but the distinction matters enough to walk through carefully.

⚠ PERHATIAN

This is not a self-directed strategy. It requires a psychiatrist or addiction medicine specialist who is actively managing the decision, monitoring for misuse risk, and treating it as a time-limited clinical tool — not an open-ended replacement. Pursuing a stimulant prescription informally, or from a general prescriber unaware of the full history, is a materially different and much riskier thing than what's described below.

The clinical logic: the Gray and Slow Return phases in Section 2 produce real, measurable executive-function impairment — attention, working memory, task initiation — that can closely resemble ADHD, whether or not ADHD was present before meth use began. In some specialist settings (this exists as documented practice in some countries' addiction medicine programs, sometimes called psychostimulant substitution or agonist-style stabilization), a carefully supervised,

long-acting prescription stimulant — extended-release amphetamine salts (Adderall XR) or similar — is used short-term specifically to bridge that executive-function gap while the dopamine system in Section 3 does its slower underlying repair work, in the same structural spirit as using buprenorphine to stabilise an opioid-dependent system rather than letting it recover entirely unsupported.

What This Is	What This Isn't
A time-limited, specialist-supervised clinical tool aimed at the specific executive-function gap in Section 2's Gray/Slow Return phases	Green light to self-medicate the crash with any stimulant, prescribed or otherwise
Used alongside, not instead of, the Tier 1 and Tier 3 work in this section	A substitute for the exercise, sleep, and neuroplasticity work that actually drives the DRD2 receptor recovery in Section 3
Evidence-limited and genuinely debated within addiction medicine — evidence tier: Emerging, not Established	A settled, first-line standard of care — most addiction specialists still reach for this cautiously, if at all

BEING HEARD

If the fog and flatness of early recovery is making you wonder whether you actually have ADHD, or always did — that's a legitimate question worth raising with a psychiatrist, not something to dismiss as 'just withdrawal talking.' Sometimes it is withdrawal. Sometimes a real, previously undiagnosed ADHD was part of what made stimulants so compelling in the first place, per Section 6's phenotyping questions. A specialist can actually help you tell the difference, which is exactly the conversation 'ghost scaffolding' is meant to open, not replace.

Tier 3 — Nervous System and Neuroplasticity Support (The Repair Signal)

- Exercise — the single most important intervention in this entire protocol. BDNF, the repair signal for the damaged dopamine axons documented in Section 2 and Section 3, is primarily generated through physical activity. Begin as soon as physically possible, even briefly, and build toward 30-45 minutes of moderate-to-high intensity most days.
- Sleep — 7-9 hours, consistent schedule; BDNF synthesis and memory consolidation both peak during sleep, meaning inconsistent sleep directly slows the repair process described in Section 3.
- Morning sunlight — 20+ minutes outside, ideally within the first two hours of waking, for circadian alignment and mood support.
- Novel, challenging learning activity — 30+ minutes daily of something actively difficult (an instrument, a language, a complex skill) drives neuroplasticity more than passive activities.
- Structured daily routine and environmental redesign — removing or altering triggers tied to use locations, people, and paraphernalia.

Section 9 — On Stigma

Methamphetamine carries a specific, heavier weight of social stigma than most other substances covered in this library — the 'tweaker' stereotype, assumptions about criminality or moral failure, and a level of disgust in how the drug and its users get talked about publicly that alcohol or even opioid dependence often doesn't carry to the same degree. This is worth naming directly rather than leaving unspoken, because it has a real, documented practical effect: people with methamphetamine use disorder report — and research on healthcare-provider bias confirms — receiving measurably worse quality care, less thorough pain management, and more dismissive treatment than from medical providers than people with other substance use disorders, even when presenting with the same symptoms.

BEING HEARD

If you've felt like you had to brace yourself before telling a doctor, a dentist, or an ER what was actually going on — that instinct isn't paranoia. It's a reasonable response to a real pattern. It doesn't mean every provider will treat you that way, and it doesn't mean you should hide the information that keeps you safe (per Section 0 and Section 5's psychosis-risk overlap, an accurate history matters for your own care) — but the caution you're carrying into that conversation is not an overreaction to nothing.

- If a provider's tone shifts noticeably once methamphetamine use is disclosed, that's worth naming directly to them ('I want to make sure this history doesn't change the quality of care I'm getting here') or, if that doesn't work, worth escalating to a different provider — you're allowed to do that.
- Addiction medicine specialists and harm-reduction-oriented clinics generally have less of this bias baked in than general practice or emergency settings — worth seeking out specifically for ongoing care where access allows.
- Stigma ni pasal reputasi ubat tu, bukan pasal awak pulak. Sistem dopamin yang dibina semula dalam Bahagian 3 tak kisah apa orang sangka pasal perangai awak masa dia buat kerja tu.

Section 10 — Emergency Recognition, Restated

⚠ PERHATIAN

Call findahelpline.com or 995 immediately for: suicidal thoughts or a specific plan, chest pain or irregular heartbeat, sudden severe headache or stroke signs (facial drooping, arm weakness, slurred speech), psychosis (paranoid delusions, hallucinations), or extreme agitation with loss of behavioral control. For the crash symptoms alone — exhaustion, hypersomnia, depression, cravings, cognitive fog — these are expected per Section 2, but a support contact and, if severe, a same-day medical or psychiatric evaluation are still appropriate.

Section 11 — Full Protocol Cross-Reference Map

Reference	What It Offers Here
Recovery Kernel Meth/Stimulant Guide, Section 3 (The Detox Protocol)	The full source timeline this guide's Section 2 is built from, plus the complete supplement dosing and timing detail behind Section 8's Tier 1.
Recovery Kernel Compendium, Section 16.4 (Receptor Recovery — The Evidence)	The Volkow et al. and Heinz et al. citations behind Section 3's 12-17 month timeline, plus the comparative data for alcohol and cocaine receptor recovery.
Recovery Kernel Compendium, Protocol 01 (Shelter)	The crisis-stabilization framework referenced throughout Section 2's crash and pit phases.
Recovery Kernel Compendium, Protocol 05 (Environmental Redesign)	Trigger-removal and environmental-modification work mentioned in Section 8, Tier 3 — identifying and changing use-related people, places, and things.
Recovery Kernel Compendium, Protocol 06 (Daily Structure)	The consistent-routine framework suitable to start from around Week 2-4 in Section 2, once the acute crash has passed.
Recovery Kernel Compendium, Protocol 20 (Identity Reconstruction) and C-28 ('The Recast')	Start appropriate around Months 1-3 in Section 2 — not earlier. Build an identity and a set of genuine, high-intensity outlets suited to the Warrior COMT profile in Section 6, rather than one organised around either the drug or its absence.
Recovery Kernel Compendium, Section 16 (Neuroplasticity Architecture)	the full 90-day rebuilding framework referenced in Section 3 — exercise, sleep, learning, and sunlight protocols in complete detail, including the weekly structure behind Section 8's Tier 3.
Recovery Kernel Chemsex Guide	If methamphetamine use occurred in a chemsex context specifically — the combined GHB/meth/mephedrone recovery timeline and unified supplement stack address the multi-substance picture this guide covers only for meth alone.
Recovery Kernel Complex Trauma and the Substance Connection	For this guide's own Section 3 (Substance-Trauma Co-Axis): chronic hypervigilance channeled into forced achievement is a documented trauma-driven pathway into stimulant use specifically — worth reading alongside this guide if that pattern applies.

Section 12 — Closing

SANCTUARY

The dopamine system that methamphetamine hijacked is not permanently broken. The data is specific and real: measurable recovery within weeks, substantial structural recovery within a year to a year and a half, and a 90-day window where deliberate exercise, sleep, and novel challenge do more rebuilding work than any other point in the process. The misery of the crash was never a sign that something had gone wrong. It was, and is, the signal that repair had already begun.

END OF GUIDE — DISCONTINUANCE PROTOCOL: METHAMPHETAMINE

Harm Reduction Resource — Not Medical Advice — Not a Substitute for Emergency Care

Crisis Resources: 988 (Suicide & Crisis Lifeline, call or text) | Crisis Text: HOME to 741741 | SAMHSA: 1-800-662-4357